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Letter to Editor
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The Ontogenes in Drosophila and the Problem of Fertility

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Letter to Editor

The human reproductive system in the area of medical knowledge is a component along with other systems even lacking its own name. It is united with the excretory system and referred to as urogenital system. Thus, it is no wonder that the disturbances of reproductive function (a decreased fertility or infertility) is considered in a manner similar to disturbance of any other biological function. Adverse environmental conditions, abnormalities of internal body regulation, somatic disorders, and genetic aberrations associated with Mendelian gene mutationsare regarded as the causes of these disturbances, for example, the genetic mutations interfering with meiosis. The current (medical, biological, and genetic) understanding of fertility of an organism is regarding it as a common biological trait, moreover, a quantitative trait. No special attention is attached to the variation in fertility in a somatically healthy organism, since just several gametes are sufficient to get several progenies. However, an exclusive role of the reproductive system in the existence of living matter is eliminated from consideration. The reproductive system is the basis of existence of the renewable living matter, while the diverse living world is only the topside on this basis. In this view, pathology of the reproductive system cannot be equated with any pathology of the other body systems. The causes of a reproductive pathology should a priori be more numerous and of another nature. The latest genetic research confirms this postulate: pathology of fertility has its unique causes. The formation of the program for individual development influences fertility. Mere "well-being" of the reproductive organs is not sufficient to guarantee the normal fertility; this requires a normal process in the preparation of individual developmental program for the progeny. Fertility depends on both the genetic events taking place during the preparation of gametes of each parent and the events occurring when the pronuclei are met in the synkaryon. Thus, the fertility level reflects both the "well-being" of the individual developmental program of each parent and their "compatibility" when they meet. Fertility plays the role of a biological regulator that interferes with genetic variation and thereby becomes a tool of evolution. The genome-wide sequencing has shown that the Mendelian genes encoding proteins cover no more than dozen percent of the genome DNA. The remaining DNA is involved in the regulatory function. A new class of mutations was discovered when studying drosophila and named *conditional mutations* [1]. We believe that the conditionalmutations are mutations of previously unknown regulatory genes. Regulatory genes control the individual development and are referred to as *ontogenes* [2]. Presumably, these particular genes occupy the above mentioned 90% of the DNA with a vague function. Ontogenes are directly associated with fertility of an individual. A mutation in an ontogene, even in a single dose, leads to the death of the progeny. Some classes of progeny fail to emerge at all, while other classes appear in a reduced number. A decrease in fertility manifests itself as a parental effect: both the progenies that received the mutation from a parent and those that have not received it die [3]. In drosophila, the progenies die in the early development, at the stage of egg. A lethal effect of a mutation in an ontogene depends on the genome of the mating partner. Depending on a particular partner, the progeny can be absent at all, be few, or close to the norm in the number and composition. The effect of the partner's genome also follows the parental effect [4,5]. We believe that ontogenes are activated during maturation of gametes in parents and the program of individual development is revisited anew. This program is encoded in terms of DNA conformation and is to be transferred with gametes. After the event of fertilization, the conformations of parental genomes are compared to make the decision on whether the development should be continued or arrested [3-5]. Thus, the fertility disturbances are caused not only by mutations in the Mendelian genes responsible for a normal function of reproductive organs, but also by mutations in ontogenes, responsible for formation of a normal program for individual development of a progeny. Then, any species is likely to have geneticvariants of the program of individual development. When meeting, such variants can lead to the death of progenies (sterility). As a rule, the mutations in ontogenes of one parent lead to its sterility; however, successful choosing of a mating pair allows for avoiding of sterility.

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